Restrained Eating Behavior and the Metabolic Response to Dietary Energy Restriction in Women

Nancy L. Keim and William F. Horn

Abstract

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Objective: To determine whether prior eating behavior characterized by dietary restraint alters responses in energy expenditure and substrate oxidation associated with a short-term, energy-restricted diet.

Research Methods and Procedures: A repeated-measures, 3-day diet-intervention study of adequate (125 kJ/kg of body weight) or restricted (62.5 kJ/kg) energy intake was conducted with 30 women, 20 to 46 years, BMI 25 to 45 kg/m^2, whose prior eating behavior was "restrained" or "unrestrained." The Eating Inventory (cognitive restraint subscale) was used to measure restrained eating behavior. Energy expenditure and substrate oxidation were measured after a 12-hour fast and during the first and fourth hours after a standard meal. Plasma glucose, nonesterified fatty acids, and insulin were measured at corresponding times. Body composition was determined by total body electrical conductivity.

Results: Resting energy expenditure was not affected by 3 days of energy restriction. Short-term energy restriction resulted in lower respiratory-exchange ratios, higher rates of fat oxidation, and lower rates of carbohydrate oxidation. Subjects classified as restrained eaters had higher postprandial respiratory-exchange ratios and carbohydrate-oxidation rates compared with unrestrained eaters. Fasting insulin concentrations were lower in restrained eaters. These effects associated with prior eating behavior were independent of the diet intervention.

Discussion: Metabolic outcomes associated with a 3-day energy-restricted diet (i.e., increased fat oxidation and decreased carbohydrate oxidation) were not affected by prior restrained eating behavior. However, restrained eating behavior was associated with increased carbohydrate oxidation after a mixed meal. This effect of restrained eating behavior may be attributable to increased insulin sensitivity.

Key words: dieting, postprandial metabolism, fat oxidation, carbohydrate oxidation, insulin

Introduction

Restriction of energy intake to a level below the energy requirement to maintain body weight is associated with a decrease in metabolic rate. Results of human studies conducted in room calorimeters indicate that even after only 1 day of consuming an energy-restricted diet, small, but significant, decreases in energy expenditure can occur (1,2), and the respiratory exchange ratio (RER)[1] also declines, indicative of a shift toward greater reliance on fat oxidation (2). In these short-term studies, it has been noted that the individual response to the energy-restricted diet intervention varied greatly. To explain some of the variation in the metabolic response to such a diet manipulation, it is interesting to consider the possibility that prior eating behavior may have an impact on energy metabolism. In previous reports, women who were classified as "restrained eaters" had reduced metabolic rate (3,4) and thermic response to a meal (5). Fat oxidation was reduced in restrained eaters when their diets were high in fat (6). These reports led us to hypothesize that prior eating behavior based on dietary restraint, or the willful tendency to limit food intake to control body weight, may impact the metabolic response to an energy-restricted diet intervention, particularly when the intervention is brief.

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Western Human Nutrition Research Center, U.S. Department of Agriculture, Agricultural Research Service, University of California, Davis, California.
Address correspondence to Nancy L. Keim, Research Nutrition Scientist, Western Human Nutrition Research Center, 1 Shields Ave., University of California, Davis, CA 95616.
E-mail: nkeim@whnrc.usda.gov

[1] Nonstandard abbreviations: RER, respiratory-exchange ratio; REE, resting energy expenditure; HOMA, homeostasis model assessment; NEFA, nonesterified fatty acid.

The current study was conducted to determine the effect of a short-term, energy-restricted diet on energy expenditure and substrate oxidation and to determine if prior restrained eating behavior influenced the metabolic response to this short (3-day) diet intervention.

Research Methods and Procedures

Subjects

Premenopausal women ($n = 30$) were recruited to participate in this study. Inclusion criteria included age 20 to 45 years, stable body weight (± 3%) for the previous 6 months, a body fat percentage >30%, self-reported regular menstrual cycles, and good health status as evaluated by medical history and examination, assessment of clinical blood chemistry panel, and resting electrocardiogram. Exclusion criteria included body weight change of more than ±3% in the previous 6 months and current or recent (within past 18 months) pregnancy. The study was reviewed and approved by the human subject committees of the University of California-Davis and the U.S. Department of Agriculture.

Study Design

A baseline evaluation of each subject included assessment of eating-behavior characteristics, resting energy expenditure (REE) after an overnight fast, and body weight and composition. The intervention consisted of a 3-day diet intervention, followed by a fourth day of metabolic testing. This sequence was repeated by each subject four times, with each intervention period scheduled ~1 month apart. For the diet intervention, subjects were randomly assigned to either an energy-restricted or an energy-adequate intervention diet and consumed this same prescribed diet for 3 days before each test session. For the metabolic testing, subjects reported to the laboratory in the morning after a 12-hour fast. Weight and body composition were measured, subjects rested quietly, REE was measured, and a fasting blood sample was obtained. A standard test meal was ingested, postprandial energy expenditure was measured for the first postprandial hour, and a 1-hour postprandial blood sample was obtained. Subjects remained in the testing area during the second and third postprandial hours. During this time, subjects were permitted to engage in light to moderate activity during the second hour and then resumed sitting quietly (watching television or reading) during the third hour. Energy expenditure was measured again during the fourth postprandial hour, and blood samples were obtained at the beginning and end of this fourth hour.

Classification of Eating Behavior

The Eating Inventory (7) was used to measure restrained eating behavior. The score for the cognitive restraint subscale was used to classify subjects as restrained eaters if the score was ≥11 or unrestrained eaters if the score was <11.

Diets

All foods and beverages in the prescribed diets were weighed and packaged for the subjects in the metabolic kitchen of the Western Human Nutrition Research Center. The food items selected for the diet required only simple preparation. Subjects prepared and consumed their meals in their homes. They were instructed to keep records of omissions and deviations from the prescribed diet.

The energy content of the 3-day diet was adjusted for each subject on the basis of her weight; the energy-adequate diet provided 125 kJ/kg of body weight/day, and the energy-restricted diet provided 62.5 kJ/kg of body weight/day. For both diets, the daily menu consisted of the same conventional foods, but different portion sizes, and was repeated for 3 consecutive days preceding the diet-intervention test day. The macronutrient composition of the two diets was the same: 60% carbohydrate, 18% protein, and 22% fat. The test meal served on the diet-intervention test day corresponded to the 3-day diet assignment and was 15% of the prescribed daily energy intake for each subject. The meal included orange juice, whole-wheat toast, margarine, grape jam, and nonfat milk. Small adjustments in the energy content of the meal were made with a complete nutrition liquid supplement (Nutrament, Mead Johnson, Princeton, NJ). The mean energy content of the test meal was 1531 ± 69 kJ for the energy-adequate diet group and 765 ± 29 kJ for the energy-restricted diet group. The macronutrient composition of the test meal was similar to that of the intervention diets: 62% carbohydrate, 18% protein, and 20% fat.

Procedures

Energy expenditure and substrate-oxidation rates were determined from respiratory gas exchange measurements (8,9). An automated metabolic cart (2900 Metabolic Cart, SensorMedics, Yorba Linda, CA) was used to determine rates of oxygen consumption (VO_2) and carbon dioxide production (VCO_2) throughout the study. Measurements of REE were taken for 30 minutes after a 12-to-14-hour fast at baseline and after the 3-day diet-intervention periods. The procedure commenced at approximately 8:00 AM. Subjects sat in a semi-reclined position in a comfortable chair, resting quietly in this position for 30 minutes before the measurement. Twenty minutes of steady-state values were averaged to determine REE. After the test meal, respiratory gases were collected and analyzed for 45 minutes during the first and fourth postprandial hour. To facilitate the collection of expired breath, subjects wore inflatable face masks that were connected to the gas-mixing chamber of the metabolic cart through a tubing assembly. The Weir equation was used to calculate energy expenditure from measurements of VO_2 and VCO_2 (8). Carbohydrate- and fat-oxidation rates were calculated using the Frayn equations (9). Urinary nitrogen excretion was estimated from the nitrogen content of the

prescribed diet, assuming that each subject was in nitrogen balance and that nitrogen was excreted at a constant rate.

Body weight was measured on arrival in the laboratory using a platform scale with a digital display (Acme Series 7000 and Ohaus PBI; Acme Scale Co., San Leandro, CA). Before being weighed, subjects voided and changed into surgical scrubs. Weight was recorded to the nearest 0.05 kg. Body composition was measured using the total body electrical conductivity method (HA-2 Body Composition Analyzer; EM Scan, Springfield, IL). The prediction equation used to estimate fat-free mass was developed specifically for overweight individuals (10).

Blood specimens were collected into vacutainers containing sodium fluoride and potassium oxalate additives. Plasma was separated by low speed centrifugation (1100*g* for 10 minutes at 0 to 4 °C) and stored at −70 °C. Glucose concentrations were determined by the hexokinase method (11) using reagents from Sigma Diagnostics (St. Louis, MO), which were adapted for use on a centrifugal analyzer (Cobas-Fara, Roche Dagnostic Systems, Somerville, NJ). Insulin concentrations were measured using a commercially available radioimmunoassay kit (Coat-A-Count; Diagnostic Products Corporation, Los Angeles, CA). Using a commercially available kit (Wako Chemicals, Richmond, VA), we determined nonesterified fatty acid (NEFA) concentrations by a colorimetric method (12). Insulin resistance was estimated by homeostasis model assessment (HOMA), using fasting values for insulin and glucose (13).

Statistical Analysis

A general linear regression model (SAS Version 6.12; SAS Institute, Cary, NC) was used to determine the effects of the diet intervention, restrained eating-behavior classification, or the interaction of these variables on energy expenditure, RER, carbohydrate- and fat-oxidation rates, and plasma concentrations of glucose, insulin, and NEFAs. Rates of energy expenditure and substrate oxidation were adjusted for differences in body size by including a covariate representative of body size in the regression model. In three separate analyses, we pretested fat-free mass, body weight to the 3/4 power, or BMI as the covariates, each yielding similar results. Because energy use occurs predominantly in the fat-free tissues and because fat-free mass has the best correlation with the energy expenditure variables, we elected to present the results of the regression analysis with fat-free mass as the covariate. Thus, all values of energy expenditure and substrate-oxidation rates are reported as least-square adjusted means and SEs. Other outcome variables, including the RER and plasma variables, were not adjusted for fat-free mass and are reported as means ±SE. The probability level for significance was set at $p < 0.05$.

Results

Subject Characteristics

Fifteen subjects were assigned to the energy-restricted diet group, 9 of whom were classified as restrained eaters and 6 as unrestrained eaters. The other 15 subjects were assigned to the energy-adequate diet group, 7 of whom were restrained eaters and 8 were unrestrained eaters. The resulting diet-intervention × eating-behavior groups were similar with respect to the Eating Inventory disinhibition and hunger scores, age, height, and body fat percentage (Table 1). The restrained eaters assigned to the energy-restricted diet group weighed less ($p < 0.05$) and had less fat-free mass ($p < 0.05$) than their unrestrained counterparts (Table 1). Body weight and body composition remained stable throughout each subject's period of enrollment in the study. (This period spanned 4 to 6 months to accommodate the repeated-measures test schedule.) Weight and fat-free mass varied by <3% within each subject during this time, averaging 1.4% and 1.2% variation, respectively, for within-subject measurements taken on the morning of the four test sessions.

Energy Expenditure

REE and fasting RER, measured at baseline before the diet intervention, did not differ according to diet-group assignment of eating-behavior classification (Table 2). After the 3-day diet intervention, REE was not different among diet-intervention × eating-behavior groups. However, REE increased from baseline in those subjects with prior restrained eating behavior (the restrained eaters) and decreased from baseline in those without prior restrained eating behavior (the unrestrained eaters) (Table 2). This main effect of eating behavior was significant ($p < 0.01$).

Postprandial energy expenditure was higher with the energy-adequate diet during the first hour than with the energy-restricted diet. This main effect of diet was significant ($p < 0.001$). The diet-group difference lessened by the fourth hour and was no longer significant (Table 3). Eating-behavior classification had no effect on postprandial energy expenditure. When either the eating-behavior classification group or the Eating Inventory cognitive-restraint score was included in the statistical model, the adjusted mean values for REE and postprandial energy expenditure of the diet groups were altered by <1%.

RER and Substrate Oxidation

Fasting RER values at baseline were similar for all groups (Table 2). Fasting RER was lower after 3 days of the energy-restricted diet ($p < 0.001$). There were no differences in intervention RER between the eating-behavior classification groups (Table 2).

RERs during the first and fourth hours after the test meal were lower with the energy-restricted diet compared with the energy-adequate diet (Table 4). Restrained eaters in the

Table 1. Dietary restraint scores and baseline physical characteristics of subjects by diet intervention group and restrained eating behavior class

Characteristic	Energy-restricted diet		Energy-adequate diet	
	Restrained ($N = 9$)	Unrestrained ($N = 6$)	Restrained ($N = 7$)	Unrestrained ($N = 8$)
Eating Inventory subscale, score (range)				
Cognitive restraint	15.2 ± 1.1^{a} (11 to 20)	5.5 ± 1.2^{b} (2 to 9)	14.4 ± 0.9^{a} (11 to 17)	5.9 ± 1.1^{b} (2 to 10)
Disinhibition	8.8 ± 1.5 (2 to 14)	9.8 ± 1.1 (7 to 15)	10.1 ± 0.9 (6 to 13)	9.4 ± 1.7 (2 to 16)
Hunger	5.1 ± 3.4 (0 to 12)	5.3 ± 2.0 (0 to 14)	6.4 ± 1.0 (2 to 10)	6.6 ± 1.2 (1 to 11)
Age, years	37.7 ± 2.3	35.5 ± 3.5	36.6 ± 3.1	33.1 ± 2.9
Weight, kg	71.4 ± 5.3^{a}	92.6 ± 5.5^{b}	$79.6 \pm 8.2^{a,b}$	$87.1 \pm 6.2^{a,b}$
Height, cm	164 ± 3	168 ± 3	166 ± 3	168 ± 4
BMI, kg/m^2	26.5 ± 1.4^{a}	32.8 ± 1.7^{b}	$28.6 \pm 2.3^{a,b}$	$30.8 \pm 2.1^{a,b}$
Fat percentage	38.9 ± 1.5	44.0 ± 1.0	41.0 ± 2.8	40.5 ± 2.0
Fat-free mass, kg	43.4 ± 2.4^{a}	51.7 ± 2.9^{b}	$45.7 \pm 2.9^{a,b}$	51.2 ± 2.7^{b}

All values are means ± SE.
Mean values with different superscripts (a and b) are significantly different, $p < 0.05$.

energy-restricted diet group had higher postprandial RERs compared with the unrestrained eaters in this diet group (Table 4).

Carbohydrate-oxidation rates were lower and fat-oxidation rates were higher in the energy-restricted diet group after the overnight fast and during the postprandial period. These main effects of diet group are depicted in Figure 1 (A and C). Eating-behavior classification affected substrate-oxidation rates during the first hour after the test meal, whereas carbohydrate-oxidation rate was ~20% higher in the restrained eaters compared with the unrestrained eaters ($p < 0.005$). This main effect of eating-behavior classification is depicted in Figure 1B. Fat-oxidation rates during the first hour were ~10% lower in the restrained eaters com-

Table 2. REE* (kJ/min) and RER† (VCO_2:VO_2) by diet intervention group and restrained eating behavior class

Parameter	Energy-restricted diet		Energy-adequate diet	
	Restrained ($N = 9$)	Unrestrained ($N = 6$)	Restrained ($N = 7$)	Unrestrained ($N = 8$)
REE				
Baseline	4.05 ± 0.14	4.09 ± 0.17	4.07 ± 0.15	4.11 ± 0.15
Intervention	4.15 ± 0.14	4.01 ± 0.16	4.22 ± 0.15	4.08 ± 0.14
Change	0.11 ± 0.07^{a}	-0.05 ± 0.08^{b}	0.16 ± 0.07^{a}	-0.07 ± 0.07^{b}
RER				
Baseline	0.83 ± 0.04	0.83 ± 0.02	0.82 ± 0.02	0.81 ± 0.02
Intervention	0.79 ± 0.01^{a}	0.78 ± 0.01^{a}	0.83 ± 0.01^{b}	0.84 ± 0.01^{b}
Change	-0.03 ± 0.01^{a}	-0.05 ± 0.02^{a}	0.01 ± 0.02^{b}	0.03 ± 0.01^{b}

* REE values are least squares means ± SE, adjusted for fat-free mass (or change in fat-free mass for REE change values).
† RER values are means ± SE.
Mean values with different superscripts (a and b) are significantly different, $p < 0.05$.

Table 3. Postprandial energy expenditure by diet intervention group and restrained eating behavior class

Time	Energy-restricted diet		Energy-adequate diet	
	Restrained ($N = 9$)	Unrestrained ($N = 6$)	Restrained ($N = 7$)	Unrestrained ($N = 8$)
First hour				
Postprandial energy expenditure*	4.92 ± 0.15^{a}	4.80 ± 0.18^{a}	5.40 ± 0.16^{b}	$5.09 \pm 0.16^{a,b}$
Increase from resting (%)†	18.6 ± 1.2^{a}	20.7 ± 2.2^{a}	27.6 ± 1.9^{b}	25.4 ± 1.8^{b}
Fourth hour				
Postprandial energy expenditure*	4.48 ± 0.13	4.59 ± 0.16	4.75 ± 0.14	4.58 ± 0.14
Increase from resting (%)†	9.2 ± 2.0	14.6 ± 2.3	12.6 ± 1.7	12.8 ± 2.2

* Postprandial energy expenditure values are least squares means ± SE, adjusted for fat-free mass.
† Increase values are means ± SE.
Mean values with different superscripts (a and b) are significantly different, $p < 0.05$.

pared with the unrestrained eaters, but this difference was not significant ($p = 0.17$). Eating-behavior classification had no effect on substrate-oxidation rates during the fourth postprandial hour.

Circulating Glucose, NEFA, and Insulin Concentrations

There were no differences in fasting glucose or insulin concentrations between the diet groups (Table 5). Fasting NEFA concentrations were higher with the energy-restricted diet compared with the energy-adequate diet ($p < 0.01$). Restrained eaters had significantly lower fasting insulin concentrations compared with the unrestrained eaters (Table 5). This difference in fasting insulin was not attributable to differences in body weight or body composition; none of these variables (body weight, BMI, fat-free mass, fat mass, or percent body fat) was significantly related to fasting insulin using analysis of covariance.

When both fasting glucose and insulin data were used to gauge insulin resistance with the HOMA model, the unrestrained eaters were more insulin resistant than the restrained eaters ($p < 0.001$). The eating-behavior classification group differences in fasting insulin and HOMA were observed for both the energy-restricted and the energy-adequate diet interventions (Table 5).

Postprandial glucose, NEFA, and insulin values are illustrated in Figure 2. One hour after the test meal, glucose values varied widely among and within the diet-intervention × eating-behavior groups, and no main effects of diet or prior eating behavior were present. NEFA values were not different among the groups. Insulin concentrations were ~50% lower in response to the energy-restricted diet ($p < 0.001$). At hours 3 and 4, insulin and glucose concentrations were similar for the diet groups, but NEFA concentrations were higher in the energy-restricted diet groups ($p < 0.001$).

Table 4. Fasting and postprandial respiratory exchange ratios by diet intervention group and restrained eating behavior class

Parameter	Energy-restricted diet		Energy-adequate diet	
	Restrained ($N = 9$)	Unrestrained ($N = 6$)	Restrained ($N = 7$)	Unrestrained ($N = 8$)
Fasting	0.79 ± 0.01^{a}	0.78 ± 0.01^{a}	0.83 ± 0.01^{b}	0.84 ± 0.01^{b}
Postprandial				
First hour	0.85 ± 0.01^{a}	0.82 ± 0.01^{b}	0.90 ± 0.01^{c}	0.89 ± 0.01^{c}
Fourth hour	0.78 ± 0.01^{a}	0.75 ± 0.01^{a}	0.81 ± 0.01^{b}	0.79 ± 0.01^{b}

Values are means ± SE.
Mean values with different superscripts (a and b) are significantly different, $p < 0.05$.

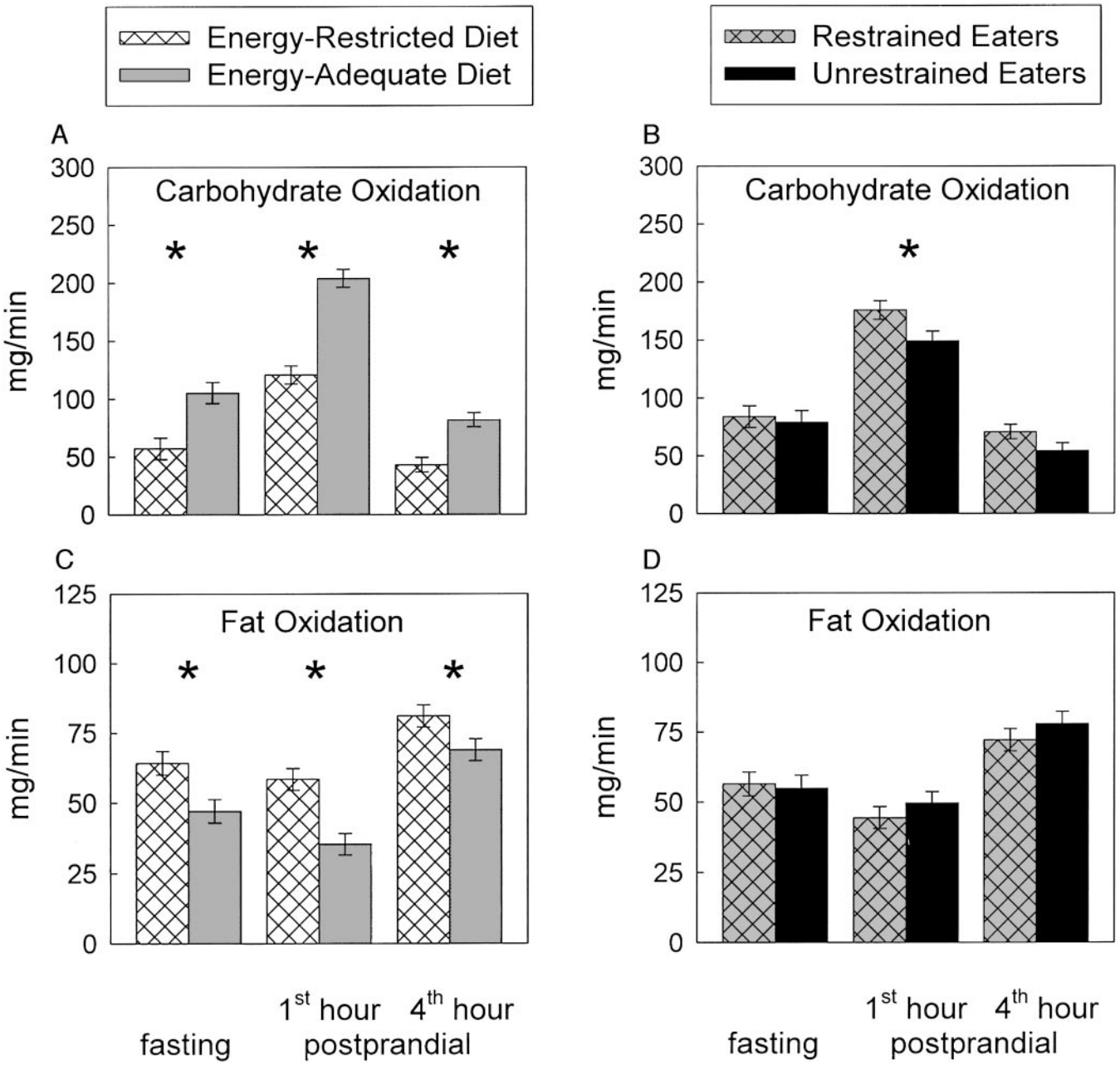

Figure 1: Carbohydrate-oxidation rates (A and B) and fat-oxidation rates (C and D) of diet-intervention groups (A and C) and eating-behavior classification groups (B and D). A and C, cross-hatched bars represent the means of the energy-restricted diet group; gray bars represent the means of the energy-adequate diet group. B and D, cross-hatched bars represent means of all prior restrained eaters; black bars represent means of all prior unrestrained eaters. All means have been adjusted for fat-free mass. Error bars represent SEs of the adjusted means. Pairs of bars marked with asterisks are significantly different ($p < 0.05$).

Discussion

Consumption of an energy-restricted diet for 3 days, with energy intake limited to 50% of that estimated to maintain weight, was not associated with lower REE. This statement is supported by the observation that intervention REE values, adjusted for fat-free mass, did not differ between the energy-restricted and energy-adequate diet groups. The lack of a diet-intervention effect on REE was not altered when eating-behavior classification was included in the statistical analysis. Thus, we were unable to confirm our hypothesis that prior dietary restraint altered the metabolic response to a dietary energy restriction. However, prior restrained eaters who consumed the energy-restricted intervention diet had increased REE compared with baseline, whereas unre-

Table 5. Fasting glucose, NEFAs, insulin, and predicted insulin resistance of subjects by diet intervention group and restrained eating behavior class

	Energy-restricted diet		Energy-adequate diet	
Parameter	**Restrained ($N = 9$)**	**Unrestrained ($N = 6$)**	**Restrained ($N = 7$)**	**Unrestrained ($N = 8$)**
Glucose (mM)	4.48 ± 0.12	4.54 ± 0.20	4.62 ± 0.15	4.75 ± 0.15
NEFA (mg/dL)	159.7 ± 11.5[a]	159.9 ± 11.6[a]	136.8 ± 9.4[b]	116.5 ± 8.7[b]
Insulin (pM)	54.6 ± 7.7[a]	85.9 ± 15.0[b]	56.2 ± 9.6[a]	88.2 ± 12.5[b]
HOMA values	1.58 ± 0.23[a]	2.43 ± 0.35[b]	1.72 ± 0.37[a]	2.71 ± 0.42[b]

Values are means ± SE.
Mean values in rows with different superscripts are significantly different, $p < 0.05$.

strained eaters consuming the energy-restricted intervention diet had decreased REE compared with baseline. This observation suggests that the plane of energy intake of the restrained eaters may have been lower during baseline when they were self-selecting their diets, compared with the habitual energy intake of the unrestrained eaters. Thus, the only evidence of an effect of restrained eating behavior on energy expenditure was the direction of change in REE when switching from the habitual diet to the controlled energy-restricted intervention diet.

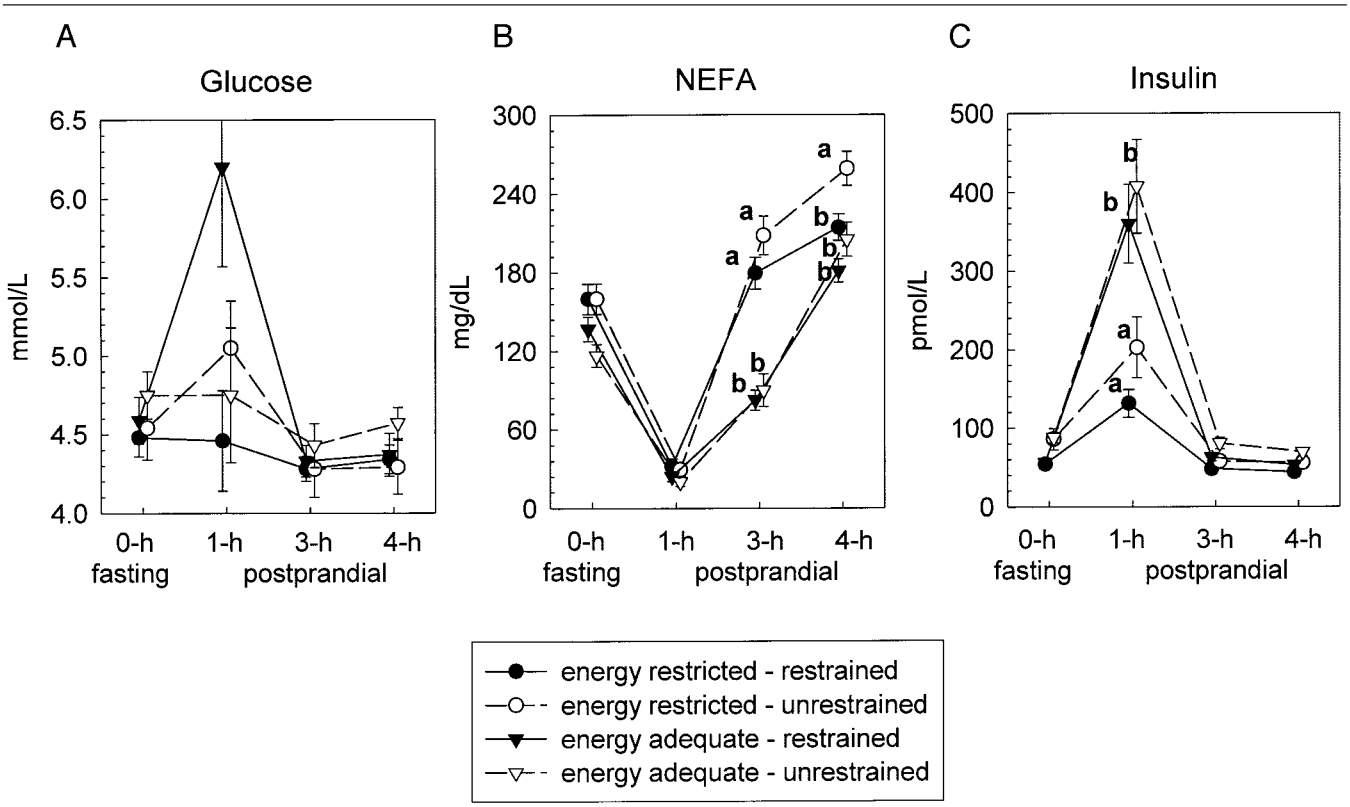

Figure 2: Circulating glucose (A), NEFA (B), and insulin (C) concentrations of diet-intervention × eating-behavior groups. Circles represent means of the energy-restricted diet groups, with filled circles indicating the subgroup of restrained eaters and open circles indicating the subgroups of unrestrained eaters; triangles represent means of the energy-adequate diet groups, with filled triangles indicating the subgroup of restrained eaters and open triangles indicating the subgroup of unrestrained eaters. Error bars are SEs. At each time-point, means marked with different letters are statistically different ($p < 0.05$).

In contrast to our energy expenditure results, dietary energy restriction has been associated with reduced REE (14,15). In previous short-term studies, energy expenditure has been shown to be reduced 6% after 1 day or 1 week of restricted intakes of 3.4 to 3.7 MJ/day (1,16). A similar reduction of 6% to 7% in sleeping energy expenditure was observed by Rumpler et al. (2) after 1 day of energy intake that was restricted to 50% of weight maintenance level. The dietary-energy restriction imposed in our study averaged ~5 MJ/day and resulted in an REE that was only 2% lower than that of the energy-adequate diet group. Several limitations of the present study may have contributed to our inability to detect differences in energy expenditure between the diet-intervention groups. Our sample size was small, with $n = 15$ per diet-intervention group. Our study design, a randomized intervention trial, made it necessary to adjust energy expenditure values for body size, a statistical maneuver that reduced between-subject variation. A crossover study design, with each subject participating in both diet interventions, would have increased the power of our study, but could have increased subject attrition rates. Finally, although we provided all food in correct portions for the diet interventions, there is a possibility that our subjects did not fully comply with their diet prescriptions. However, we did find evidence of compliance: there were clear differences in fasting RERs such that the energy-restricted diet intervention led to a decreased fasting RER compared with baseline RER values and to intervention RER values measured in the energy-adequate diet group.

We did observe different rates of energy expenditure between the energy-restricted and energy-adequate diet groups in the first postprandial hour when energy expenditure was 7% lower ($p = 0.02$) with the restricted diet. This result was expected because the energy-adequate diet group consumed a test meal that was twice as large as that of the energy-restricted group (1.5 MJ vs. 0.76 MJ). However, again, we observed that neither inclusion nor exclusion of the eating-behavior classification in the statistical model altered this acute diet effect on postprandial energy expenditure.

The effect of restrained eating behavior on energy metabolism is controversial, and only a few studies have examined energy expenditure or metabolic variables in restrained eaters. Using the doubly labeled water technique for measuring 24-hour energy expenditure, Tuschl et al. (3) found that the daily energy expenditure of women who chronically restrained their food intake was ~2.6 MJ per day lower than that of unrestrained controls, suggesting that their energy requirement for maintaining body weight was lower. Poehlman et al. (4) also reported that the resting metabolic rate, adjusted for fat free mass, was inversely related to dietary restraint in women. Platte et al. (17) found that resting metabolic rate was reduced significantly in restrained eaters. It should be noted that, similar to our findings, other investigators have failed to find an effect of restrained eating behavior on resting metabolic rate (18,19) or the thermic effect of food (17,18).

The use of specific energy substrates may be altered in response to weight loss resulting from dietary energy restriction. Astrup et al. (20) observed that the ratio of fat to carbohydrate oxidation was lower in postobese women, and Ranneries et al. (21) determined that the apparent preference for carbohydrate fuels in postobese women was a result of a decreased ability to oxidize fat, because the women had normal rates of fat mobilization and higher levels of circulating NEFAs. In the postprandial state, Raben et al. (6) found that fat oxidation was suppressed in formerly obese women after ingestion of a high-fat meal.

Restrained eating behavior has also been linked to altered substrate use. Laessle et al. (22) reported that normal-weight, restrained women had significantly higher concentrations of fasting plasma triglycerides compared with unrestrained women, suggesting that mobilized fatty acids are more readily esterified in chronically restrained eaters.

With regard to substrate use, we found that consumption of a short-term energy-restricted diet affected the use of energy, as estimated by RER and calculated fat- and carbohydrate-oxidation rates. The increase in fat oxidation and the decrease in carbohydrate oxidation with the energy-restricted diet were expected, because others have reported a rapid change in substrate oxidation with short-term energy restriction (1,2). Furthermore, we found no evidence to indicate that prior restrained eating behavior modulated the fasting substrate use response to the short-term diet energy-restriction. We did, however, find that in the first hour after the ingestion of a high-carbohydrate meal, the oxidation of carbohydrate was enhanced in those subjects with prior restrained eating behavior. This behavior effect on carbohydrate-oxidation rate was independent of the diet-intervention effect. Verboeket-van de Venne et al. (23) found that 24-hour respiratory quotient and carbohydrate oxidation were higher and fat oxidation lower in restrained subjects. The restrained eating-behavior effect on fat oxidation was accentuated when low carbohydrate diets were consumed. In our study, despite the observed differences in postprandial RER between eating-behavior classification groups, fat oxidation was not significantly altered by prior restraint. We might have observed a stronger effect of restrained eating behavior on fat oxidation if our test meals had been comprised of less carbohydrate and more fat.

Insulin plays a predominant role in energy fuel use. We observed that the restrained eaters had reduced fasting insulin levels compared with the unrestrained eaters. This finding was independent of the diet-group assignment or differences in body weight or composition. When the HOMA technique was applied, our restrained eaters were also less insulin resistant. Pirke et al. (24) reported significantly lower fasting insulin levels in restrained eaters of normal body weight. Similarly, Raben et al. (6) found

evidence of increased insulin sensitivity in formerly obese women predisposed to obesity. Taken together, these observations lend support to the idea that insulin sensitivity may be increased in individuals with restrained eating behavior. With this increased insulin sensitivity, it would be expected, as we observed, that carbohydrate oxidation is favored in restrained eaters.

In conclusion, carbohydrate oxidation was increased early in the postprandial period in women with prior restrained eating behavior. This finding may be attributable to increased insulin sensitivity in this group. Restrained eating behavior is an important element that deserves consideration in future studies of postprandial metabolism. Elevated RER and reduced propensity to oxidize fat, which are also characteristic of postobese subjects (6,21,25), may be indicative of a metabolism that favors storage of fat and have been linked to increased risk of weight gain (26). A better understanding of the biochemical factors underlying this altered metabolism in individuals with restrained eating behavior and in postobese subjects is needed.

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